Thymic hyperplasia is frequent (38% of cases) in patients with Graves’ disease (GD) but it rarely is large enough to be detected radiologically as an anterior mediastinal mass. In the few operated cases, lymphoid hyperplasia (i.e. lymphoid follicle proliferation with expansion of both the cortical and the medullary thymus component) has been documented histologically in most of the cases, while true thymic hyperplasia, i.e. thymic enlargement with normal tissular architecture, was found more rarely. In only 4 out of 107 patients with GD and thymic enlargement a malignant tumor was reported (thymoma or lymphoma/leukemia). The mechanisms of this association remain unclear, both autoimmunity and hyperthyroidism being a potential cause. The thyrotropin receptor has been identified in the thymus and may be stimulated by the TSH-receptor antibodies. Hyperthyroidism persists after thymectomy but the treatment of hyperthyroidism with antithyroid drugs usually results in a decrease of the thymus over 3-6 months.

**Introduction**

To describe the evolution of 3 patients with GD and thymic enlargement detected incidentally on CT scan. In 2 patients, a 49 years old female and a 28 years old male, the thymic mass (3.3/1.6 cm and 5.5/2.5 cm, respectively) shranked to normal after 4 and 6 months of treatment with methimazole (MTZ). In a 37 years old female, the thymic mass 4.8/3.7 cm persisted after 5 months of MTZ and was operated.

**Objective**

To describe the evolution of 3 patients with GD and thymic enlargement detected incidentally on CT scan.

**Results**

**Patient 2, male, 28 years old**

Known with complete situs inversus, diagnosed with GD, bilateral mild exophthalmos and thymic hypertrophy occupying all the thymic lodge (CT done for persistent cough).

At diagnosis, serum TSH = 0 mU/L, FT4 = 3.99 ng/dL, T3 = 6.65 ng/mL, TRAB = 40 IU/L and TPOAb >1000 IU/mL. Treated with methimazole (40 mg/day initially) and oral methylprednisolone. After 4 months of MTZ and 2 months of oral methylprednisolone in progressively lowered doses, there was a significant shrinkage of the thymus mass from 5.58/2.55/4.48 (Fig. 1) to 1.3/2.9/2.9 cm (Fig 2).

**Patient 3, female, 37 years old**

GD in April 2014 (TSH=0.005 mmol/L, FT4=1.9 ug/dL, N=0.9-1.71), TPOAb 107.8 Ul/mL (N=34), small goiter, no exophthalmos, treated with 5-10 mg/day MTZ until Sep 2014 (when still hyperthyroid), then 15 mg/d, thyroid hormones normalized in Nov 2014. July 2014 - pneumonia, CT: homogenous thymic mass 4.8/3.7 cm, lymph nodes in mediastinum, R hilum and axillae (Fig 3). Dec 2014 CT: homogenous stable thymic mass 3.1/4.1/3.9 cm (Fig 4), stable lymph nodes in the axillae.

**Conclusions**

- When a thymic mass is radiologically detected in association with Graves’ disease, in the absence of myasthenia gravis and/or suspect CT findings (nonhomogeneity, invasion in the surrounding tissues, calcification, septum, cystic lesion), it usually is thymic hyperplasia.
- Only antithyroid treatment and radiological follow-up are usually necessary.
- By recognizing the association between thymic hyperplasia and GD and the benign course of thymic hypertrophy after hyperthyroidism treatment, a major surgical procedure for a thymus mass, with potential risks, can be avoided.

**References**

2 V. Desforges-Bullet et al., Annales d’Endocrinologie 2011; 72: 304–309
3 Boyd JD, Juskevicius R. Thyroid Research 2012, 5:5